Conferences and Reviews

Hemobilia—Evolution of Current Diagnosis and Treatment

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"Hemobilia," upper gastrointestinal tract bleeding that originates from within the biliary tract, has become widely recognized due to an increased clinical awareness of the disorder and to improvements in diagnostic techniques. In addition, the growing use of percutaneous liver puncture for the diagnosis of and therapy for hepatobiliary diseases and the increased incidence of both blunt and penetrating hepatic trauma have contributed to a rising incidence of hemobilia. We review the history, pathophysiology, and current approaches to the diagnosis and treatment of this disease.

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In his extensive review of hemobilia in 1972,[1] Sandblom credits Francis Glisson with the first reported case of hemobilia.[2] In 1654, Glisson described the clinical course of a young nobleman who was stabbed with a sword in the right upper quadrant and died following massive upper gastrointestinal hemorrhage. At postmortem examination, the young man's bleeding was found to originate from a deep laceration of the liver. Glisson used his knowledge of liver anatomy to propose a pathogenetic mechanism for the bleeding: "[T]here is no doubt that the bile ducts take unto themselves (to the great good of the patient) blood issuing from the liver. From there it is either impelled upwards through reverse peristalsis, or downwards in the normal way."[2]

Scattered case reports of biliary tract hemorrhage appeared over the next 100 years, but all were diagnosed postmortem.[1] In 1777, Antoine Portal was the first to recognize a case of hemobilia antemortem.[3] The vigorous application of leeches and serial bloodlettings were unsuccessful, and the patient died. Quincke described a case of hemobilia in 1871, emphasizing the characteristic clinical features of right upper quadrant pain, jaundice, and upper gastrointestinal tract bleeding. These signs and symptoms are now recognized by modern authors as the components of the classic clinical "triad."[4]

The first surgical approach to hemobilia was reported in 1895, when a total gastrectomy was done but was not effective in controlling the patient's hemorrhage. The patient died five days later.[5] This was the first of numerous inappropriate surgical procedures performed for hemobilia. Surgeons have attempted such procedures as cholecystectomy, antireflux procedures, fecal diversion, and colon resection in an attempt to stop the bleeding of hemobilia, with predictably bad results.[1] The imprecision of diagnostic techniques in the distant past led to misguided operative decisions made in the face of threatened exsanguination.

In the first successful (and appropriate) operation for hemobilia, Kehr in 1903 identified an aneurysm of the right hepatic artery that had ruptured into the gallbladder neck, and he treated the lesion by direct arterial ligation.[6] The first successful hepatic resection for hemobilia was not reported until 1957.[7] This hepatic resection was done only after a gastric resection and subsequent cholecystectomy had failed to control the patient's hemorrhage!

The term "hemobilia" was first coined by Sandblom in his 1948 paper entitled "Hemorrhage into the Biliary Tract Following Trauma: Traumatic Hemobilia."[8] In 1971, Sandblom published his collected works on hemobilia in the first and only book devoted entirely to this subject.[1] A few years later, Walter was the first to use angiographic embolization for the treatment of this disease,[9] a modality that has since become the mainstay of treatment in most cases of hemobilia.[10-13]

Etiology

Hemobilia may occur in a variety of settings, including trauma, cholelithiasis, acalculous inflammatory diseases, vascular disorders, and neoplasms, in descending order of reported frequency (Table 1).[1,14,15]

Posttraumatic hemobilia is the most common etiologic category, accounting for 55% of cases in Sandblom's collected series[1] and 40% to 85% of cases in more recent reports that reflect a rise in the incidence of blunt and penetrating liver injury due to violent crime and vehicular accidents and the increased use of percutaneous biopsy and intubation techniques for managing hepatobiliary diseases.[13-15] The liver is the most commonly injured abdominal organ,[16] and hemobilia may complicate the clinical course of as many as 3% of patients with substantial liver injuries.[1,14,17,18] In two recent

TABLE 1.—*Causes of Hemobilia*

Cause	Patients, No. (%), by Source			
	Sandblom (n=355)*	*Curet† (n=86)*	*Yoshida‡ (n=103)*	*Collected Series (n=544)*
Iatrogenic trauma	59 (17)	50 (58)	42 (41)	151 (28)
Accidental trauma	137 (38)	23 (27)	20 (19)	180 (33)
Gallstones	53 (15)	--	9 (9)	62 (11)
Acalculous inflammation	46 (13)	--	10 (10)	56 (10)
Vascular conditions	38 (11)	--	15 (14)	53 (10)
Neoplastic disease	22 (6)	--	7 (7)	29 (5)
Unspecified atraumatic	--	13 (15)	--	13 (2)

*From Sandblom.[1]
†From Curet et al.[14]
‡From Yoshida et al.[15]

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series, 83% to 95% of cases of traumatic hemobilia occurred after blunt injury.[14,15] This finding is likely due to the fact that penetrating injuries are usually explored primarily at the time of injury and adequately treated at the initial operation. Posttraumatic bile stasis, hematomas, and abscesses all predispose to the development of hemobilia. Based on the experience with children who have sustained trauma,[17] there is a trend toward the initial nonoperative management of blunt injuries in adults, and one might expect an increased incidence of hemobilia with a more widespread application of this strategy.

Iatrogenic hemobilia may occur from percutaneous liver procedures, liver or biliary operations, or therapeutic anticoagulation. The use of percutaneous liver puncture for biopsy, cholangiography, and drainage of the biliary tract has become common, sparing many patients open surgical procedures for the diagnosis and management of some hepatic diseases.[13-15,19-24] These procedures, however, are the equivalent of a penetrating liver injury that is treated nonoperatively. Given the close proximity of bile duct radicals to the branches of the hepatic artery and portal vein, the substantial incidence of concurrent injury to these structures and fistula formation is not unexpected. A 3.8% incidence of hepatic vascular abnormalities was found following percutaneous transhepatic cholangiography, a 5.4% incidence of hepatic vascular abnormalities following percutaneous liver biopsy, and a 26.2% incidence following the placement of indwelling transhepatic drainage catheters.[25] These findings were confirmed in 1980 in patients with drainage catheters.[26] The frequency of clinical hemobilia ranges from less than 1% for liver biopsy[19,20] to 4% for transhepatic cholangiography[23] and 3% to 14% for percutaneous transhepatic catheter drainage.[21,22] The incidence appears to increase with larger caliber instruments, central liver punctures, and indwelling catheters (which may cause pressure necrosis of adjacent blood vessels). Iatrogenic hemobilia may also occur after operations on the liver or biliary tract,[1,13,24,27,28] and there is a single report of iatrogenic hemobilia occurring in a series of patients following the placement of indwelling hepatic artery catheters.[27] Finally, biliary bleeding has been reported as one of the iatrogenic complications of therapeutic anticoagulation.[24]

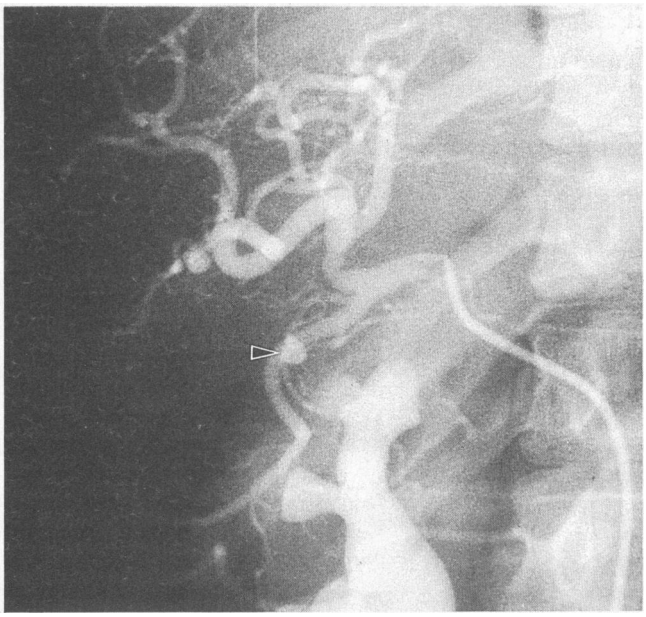

Figure 1.—This patient had hemobilia develop after a common bile duct exploration. Her celiac angiogram showed a pseudoaneurysm in the gastroduodenal artery **(arrow)**, but the location was felt to be unsafe for embolization because of technical problems encountered while attempting to occlude the vessel. The pseudoaneurysm was ligated during celiotomy, and the patient has had no recurrent bleeding (case courtesy of Richard Price, MD, and Robert Ghelfi, MD).

Upper gastrointestinal tract bleeding due to gallstones accounts for 9% to 15% of all reported cases of hemobilia.[1,15] Unlike in posttraumatic cases, bleeding in this setting is more likely to be minor. Occult rectal bleeding classically was thought to be a physical sign of cholelithiasis and may occur in as many as 37% of all patients with gallstones.[1,29] Hemorrhage may result from direct mucosal erosion or from hemorrhagic necrosis complicating severe cholecystitis or cholangitis.[13,29]

Acalculous inflammatory conditions of the biliary tract are implicated in 10% to 13% of reported hemobilia cases.[1,15] This cause accounts for a higher proportion of cases in underdeveloped countries, where parasitic diseases are more prevalent and violent crime or high-speed blunt trauma are relatively less common. The leading cause of hemobilia in this category is ascariasis, a disease with an estimated worldwide prevalence of 25% and a striking propensity to infest the biliary tract.[30] Hemobilia may also complicate the clinical course of hepatic abscess, acalculous cholecystitis, and cholangitis.[1,31,32] Rare cases of biliary hemorrhage have been reported with hepatitis, pancreatitis, and chemical inflammation or erosion due to heterotopic stomach.[1,33]

Primary vascular causes of hemobilia have been identified in 11% to 15% of cases.[1,15] The differential diagnosis in this area includes aneurysmal disease of the hepatic artery or portal vein or vasculitis.[1,7,13,34-36] Portal hypertension has traditionally been cited as a vascular cause of hemobilia,[1] but only five cases of spontaneous biliary hemorrhage from the portal circulation have been reported,[36-38(p102),39] and all but one originated from extrahepatic sources.[39] Portal hypertension is more likely to be a predisposing factor for bleeding after accidental or iatrogenic trauma rather than a primary cause.[34,39] Biliary hemorrhage from vascular sources tends to be massive and life-threatening.

The last major cause of hemobilia is neoplastic disease, accounting for 6% of cases reported.[1,15] Most cases occur because of hepatocellular carcinoma, although fibrolamellar hepatoma, hemangiomas, cholangiocarcinoma, gallbladder carcinoma, and even adenocarcinoma of the pancreas may rarely cause biliary hemorrhage.[1,40,41] Benign lesions of the bile ducts and gallbladder and tumors metastatic to the liver or biliary tract are also rarely associated with hemobilia.[1,42-44] Hemobilia from these sources tends to present as chronic anemia.

Diagnosis

The clinical presentation of hemobilia includes biliary colic in 70% of patients, jaundice in 60%, and gastrointestinal bleeding in all patients, although this may range from occult to massive bleeding.[1] The classic triad of pain, jaundice, and bleeding is present in only 32% to 40% of patients.[13-15] Yoshida and colleagues reported that the initial diagnosis was made with angiography (28%), endoscopy (12%), intraoperatively (34%), and with the noting of blood in various biliary drains (12%).[15] Anecdotal reports indicate that technetium-labeled erythrocyte scans and the presence

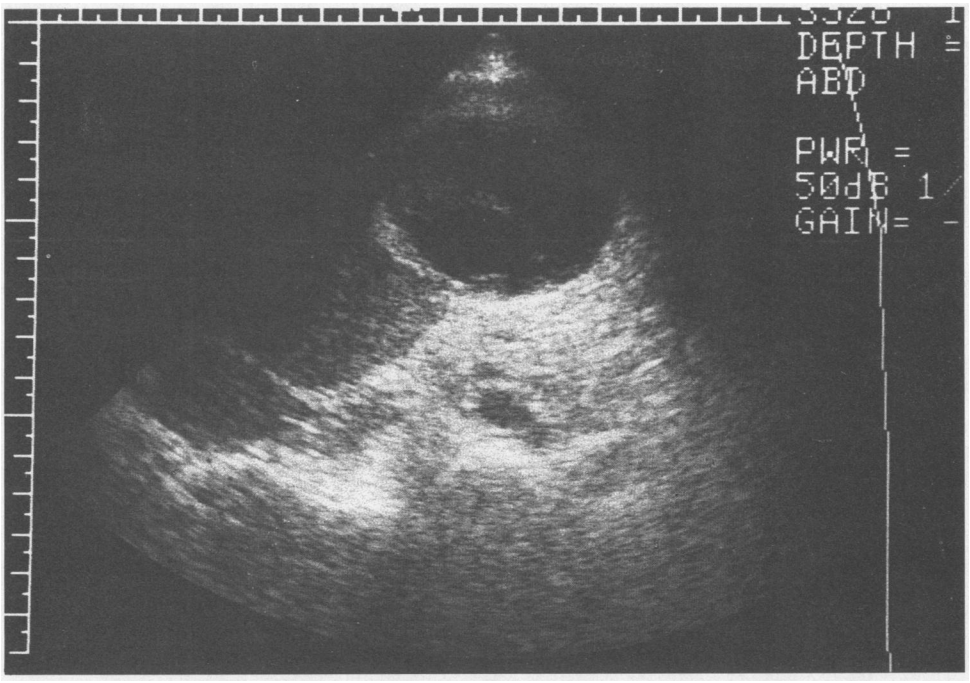

Figure 2.—An ultrasonogram shows a cystic mass in the gallbladder, which was the only abnormality identified during preoperative evaluation of a patient with hemobilia. The patient underwent cholecystectomy, but the cystic mass was an organized blood clot. The patient was later found to have a hepatic source for the hemobilia (reprinted with permission from Merrell et al[39]).

of fecal occult blood occasionally support or suggest the diagnosis,[45] but a reliance on these tests for diagnosis cannot be justified.

Angiography is the most useful diagnostic and therapeutic modality, but it is not always successful (Figure 1).[9-15,46] The variable rate and intermittent nature of bleeding may hinder diagnosis. Furthermore, anomalous, atherosclerotic, or tortuous vessels or a previous hepatic artery ligation may limit access to the arterial supply of the liver or bile ducts.[13] The chief virtue of angiography is that diagnosis can be combined with therapeutic embolization, the treatment of choice for most cases of hemobilia.

Other methods of diagnosis may include computed tomography and ultrasonography. Computed tomography is useful in cases of liver trauma and may help identify hematomas and bile collections or deep parenchymal disruptions that could predispose to hemobilia.[14,16,17] Ultrasonography may also be helpful in the diagnosis of hemobilia, but it is nonspecific. Abnormalities seen on ultrasound examinations may or may not be causing hemobilia or may be the result of hemobilia (for instance, blood clots in the gallbladder) (Figure 2).[14,39,47,48]

Hemobilia is more frequently being diagnosed endoscopically.[15,27,49,50] Although only 12% of cases are initially diagnosed with endoscopy, it may confirm the diagnosis in an additional 30% of patients and help exclude other causes of upper gastrointestinal tract bleeding.[15,27] Endoscopic access to the biliary system through percutaneous tube tracts has also been successfully used for the laser coagulation of biliary bleeding.[51] With the development of biliary endoscopes, the increasing use of nasobiliary catheters, and endoscopic sphincterotomy, endoscopic procedures may become increasingly important in the diagnosis and therapeutic management of hemobilia.[50]

If all of these measures fail to provide a diagnosis, or if the patient presents under emergency circumstances with hemodynamic instability, the surgeon may be forced to do an exploration without a precise preoperative diagnosis. Active bleeding from the duodenum may be apparent following a gastrotomy or may be detected by a rapid filling of the proximal small bowel after portioning into segments with Penrose drain "tourniquets."[39] Occasionally a lateral duodenotomy may show the expulsion of vermiform clots from the ampulla. Finally, intraoperative cholangiograms may show filling defects in the bile ducts and confirm the diagnosis (Figure 3).

Pathophysiology

A spectrum of presentations is possible: Bleeding may be either scanty or profuse, prolonged or brief, continuous or intermittent. Hemobilia may arise from any anatomic site that communicates with the biliary system (Table 2).[1,14,19] The fate of clots in the biliary tree may involve one of four possibilities: dissolution, spontaneous expulsion from the biliary tree, a combination of dissolution and expulsion, or persistence within the biliary system.[1,27,52] Acute biliary obstruction by clot may cause cholangitis, cholecystitis, or pancreatitis.[1,32] Also, symptomatic hemobilia due to biliary obstruction may result not only from blood clots but from fibrin clots, or leukocyte-fibrin clots. Furthermore, clot that remains in the bile ducts for long periods of time may serve as a nidus for gallstone formation.[53]

Through several simple but elegant experiments that were correlated with careful clinical observations, the differences in biochemical characteristics of bile and blood and the influ-

TABLE 2.—*Anatomic Origin of Hemobilia*

Site of Origin	Patients, No. (%), by Source: Sandblom* (n=355)	Yoshida† (n=103)	Collected Series (n=458)
Liver	187 (53)	70 (68)	257 (56)
Extrahepatic bile ducts	80 (22)	22 (21)	102 (22)
Gallbladder	82 (23)	9 (9)	91 (20)
Pancreas	6 (2)	2 (2)	8 (2)

*From Sandblom.[1]
†From Yoshida et al.[15]

ence of bile on wound healing in the liver were studied.[52,54] In the course of these experiments, blood and bile were found to have different specific gravities: 1.005 and 1.010, respectively. Measurements of surface tension disclosed that the surface tension of blood is 60 dynes per cm compared with 40 dynes per cm for bile. This difference is sufficient to allow a stable interface to form under some circumstances.[51] In a series of experiments using an in vitro model of the biliary tree, Sandblom and co-workers were able to simulate the conditions of blood mixing in bile and postulated mechanisms for the genesis of biliary symptoms related to hemobilia.[29,55] The rapid mixing of blood with bile created a dispersion in which no discrete clots developed. The resultant coagulum was characterized as "mushy" and "sticky." In contrast, during the slow injection of fresh blood into flowing bile, there was no mixing and pure clots formed. The differing specific gravities and surface tensions of blood and bile likely accounted for this phenomenon. In addition, clot organization resulted in an additional decrease in blood specific gravity, allowing clots to float.

In studies of the fibrinolytic properties of bile in vitro, clot lysis occurred only with warm bile, which suggested an enzymatic mechanism. It was also found that clot lysis required bile flow past the clot. This flow dependence of fibrinolysis was confirmed in an animal model.[52]

In experiments on the healing of liver wounds, fibrin production, granulation tissue formation, and scarring were diminished in the presence of bile.[54] Sandblom and colleagues postulated that the exposure of liver injuries to pooled bile slowed overall wound healing and prolonged the risk period for the genesis of hemobilia.

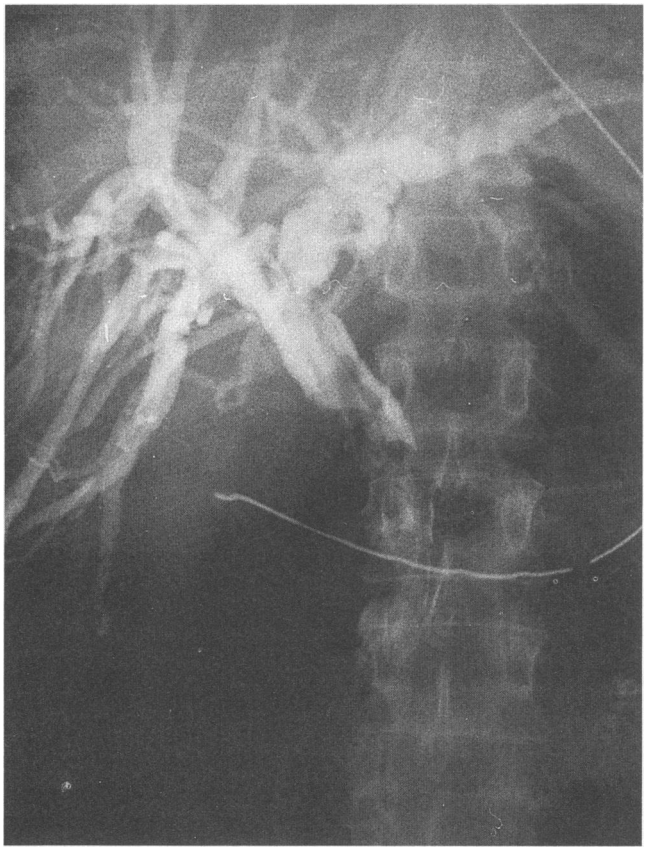

Figure 3.—A transduodenal cholangiogram shows many filling defects in the common bile duct with proximal ductal dilatation. The filling defects represent blood clots from hepatic hemobilia (reprinted with permission from Merrell et al[39]).

Several clinical correlates of these experimental findings were recognized. Slow bleeding was more likely to be associated with the formation of durable clots and biliary obstruction. Because clot lysis requires bile flow, biliary diversion through a T tube, in the presence of ampullary stenosis, contributed to distal common duct stasis and clot persistence. Filling defects seen on cholangiograms could well represent blood clots rather than stones, especially if they were floating.[55] It was thought that clots have the potential to cause late stone formation as a result of pigment deposition, encrustation, or calcification. Finally, ensuring adequate biliary tract drainage or the drainage of liver lacerations following hepatic trauma could decrease bile pooling, promote healing, and reduce the risk of hemobilia.

Therapy

The goals of therapy in cases of hemobilia are to stop the bleeding and to restore bile flow past clots. Modalities used to stop bleeding include angiography with embolization, surgical intervention, observation, and electrocoagulation or photocoagulation. Angiography is clearly the most efficacious method for controlling intrahepatic bleeding sources, with success rates above 95%.[11-15,46] In the past, the successful embolization of extrahepatic bleeding sites was hampered by the technical inability to gain sufficiently selective arterial access, leading to complications caused by nonselective embolization. For anatomic reasons, most authors have advocated surgical therapy for extrahepatic hemobilia. More recently, angiographic embolization has been shown to be useful for the control of extrahepatic bleeding even after surgical failure.[15] Some causes of extrahepatic bleeding may lend themselves to surgical correction such as bile duct tumors or bleeding from the gallbladder mucosa. Therefore, the cause of bleeding should be considered in planning therapy. But technical features of such cases, such as scarring from a previous operation, may limit surgical effectiveness. Thus, the specific anatomy should be carefully evaluated in cases of hemobilia from extrahepatic sites, and embolization should be considered when it appears the safest method and technically possible.[13]

Although surgical treatment is highly successful in controlling intrahepatic hemobilia after failed transcatheter embolization, it does not approach the success rate of transcatheter embolization as the initial treatment.[15] In one series, surgical treatment led to the successful control of 77% of hemobilia cases when hemobilia arose from extrahepatic sources.[15] Angiographic techniques, however, were successful in two cases of extrahepatic hemobilia when it was attempted as initial therapy. This confirms that embolization is useful in the management of selected patients with extrahepatic hemobilia when vessel ligation fails.

Surgical therapy may be most helpful in cases of hemobilia after blunt liver trauma for which debridement, drainage, and vessel ligation are important.[1,27] Studies of liver healing in patients with substantial hepatic trauma would support the placement of drains to reduce the possibility of bile pooling.[54] The performance of selective hepatic artery ligation is a surgical maneuver that may be useful in occasional hemobilia cases.[13] It is interesting to note that hemobilia from a portal venous source, though exceedingly rare, is likely to require surgical treatment.[28] Finally, surgical therapy should

be considered the treatment of choice when the underlying cause of hemobilia constitutes an independent indication for such treatment, such as cases associated with cholelithiasis, cholecystitis, or resectable neoplasms. Although an operation carries an obvious risk of morbidity, transcatheter embolization is not without substantial risk. Hepatobiliary necrosis (6%), abscess formation (9%), bleeding (6%), and gallbladder fibrosis (2%) have occurred following arterial embolization.[11-15]

The last commonly used therapeutic option for managing hemobilia is that of expectant observation. Spontaneous cessation of bleeding occurs most commonly in patients who undergo percutaneous cholangiography or liver biopsy; therefore, this group merits observation as primary management. Some authors have proposed the prophylactic administration of clot promoters such as absorbable gelatin sponges (Gelfoam, Upjohn, Kalamazoo, Michigan) into percutaneous puncture tracts during withdrawal of the instruments or drains from the liver as a means of reducing the frequency of bleeding complications.[25,26]

Endoscopic techniques for controlling hemorrhage and managing clots include nasobiliary drainage, endoscopic sphincterotomy, and laser photocoagulation using small endoscopes placed through a catheter tract with access to the biliary tree.[15,49-51] These methods have only been reported anecdotally and will probably continue to have a role in a selected few patients.

Summary

The successful diagnosis of hemobilia depends on a high index of suspicion, especially when the common causes of upper gastrointestinal tract hemorrhage have been excluded. A clinical suspicion should prompt confirmation of the diagnosis with angiography or endoscopy, but computed tomography or ultrasonography may also be helpful. Careful consideration of the specific anatomy will help define the therapeutic options—angiography versus surgical intervention. If access is available to the biliary tree through a percutaneous tube tract, the possibility for successful electrocoagulation or photocoagulation exists using biliary endoscopy. In occasional cases, all preoperative studies may be negative, and surgical exploration may provide the only diagnostic and therapeutic option.

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